What is claimed is:

1. A soluble CTLA4 mutant molecule having the extracellular domain of CTLA4 which binds CD80 or CD86.

2. The soluble CTLA4 mutant molecule of claim 1, wherein the extracellular domain of CTLA4 has one or more mutations in a region S25-R33 of CTLA4, and wherein the mutation is a substitution of any amino acid of serine at +25 through lysine at +28 with a different amino acid selected from alanine, arginine, asparagine, aspartic acid, cysteine, glutamine, glutamic acid, glycine, histidine, isoleucine, leucine, lysine, methionine, phenylalanine, proline, serine, threonine, tryptophan, tyrosine, and valine.

3. The soluble CTLA4 mutant molecule of claim 1, wherein the extracellular domain of CTLA4 has one or more mutations in a region S25-R33 of CTLA4, and wherein the mutation is a substitution of alanine at position +29 with any of arginine, asparagine, aspartic acid, cysteine, glutamine, glutamic acid, glycine, histidine, isoleucine, leucine, lysine, methionine, phenylalanine, proline, serine, threonine, tryptophan, or valine.

4. The soluble CTLA4 mutant molecule of claim 1, wherein the extracellular domain of CTLA4 has one or more mutations in a region S25-R33 of CTLA4, and wherein the mutation is a substitution of any amino acid of threonine at +30 through arginine at +33 with a different amino acid selected from alanine, arginine, asparagine, aspartic acid, cysteine, glutamine, glutamic acid, glycine, histidine, isoleucine, leucine, lysine, methionine, phenylalanine, proline, serine, threonine, tryptophan, tyrosine, and valine.

5. The soluble CTLA4 mutant molecule of claim 1, wherein the extracellular domain of CTLA4 has one or more mutations in a region E95-G107 of CTLA4, and wherein the mutation is a substitution of any amino acid of

glutamic acid at +95 through lysine at +96 with a different amino acid selected from alanine, arginine, asparagine, aspartic acid, cysteine, glutamine, glutamic acid, glycine, histidine, isoleucine, leucine, lysine, methionine, phenylalanine, proline, serine, threonine, tryptophan, tyrosine, and valine.

6. The soluble CTLA4 mutant molecule of claim 1, wherein the extracellular domain of CTLA4 has one or more mutations in a region E95-G107 of CTLA4, and wherein the mutation is a substitution of any amino acid of methionine at +97 through tyrosine at +103 with a different amino acid selected from alanine, arginine, asparagine, aspartic acid, cysteine, glutamine, glutamic acid, glycine, histidine, isoleucine, leucine, lysine, methionine, phenylalanine, proline, serine, threonine, tryptophan, tyrosine, and valine.

7. The soluble CTLA4 mutant molecule of claim 5, wherein the mutation is a substitution of tyrosine at position +103 with a different amino acid selected from a group consisting of arginine, asparagine, aspartic acid, cysteine, glutamine, glutamic acid, glycine, histidine, isoleucine, leucine, lysine, methionine, phenylalanine, proline, serine, threonine, tryptophan, tyrosine, and valine.

8. The soluble CTLA4 mutant molecule of claim 1, wherein the extracellular domain of CTLA4 has one or more mutations in a region E95-G107 of CTLA4, and wherein the mutation is a substitution of any amino acid of leucine at +104 through glycine at +107 with a different amino acid selected from alanine, arginine, asparagine, aspartic acid, cysteine, glutamine, glutamic acid, glycine, histidine, isoleucine, leucine, lysine, methionine, phenylalanine, proline, serine, threonine, tryptophan, tyrosine, and valine.

9. The soluble CTLA4 mutant molecule of claim 1, wherein the extracellular domain of CTLA4 has one or more mutations in a region beginning with

asparagine at position +108 and ending at isoleucine at position +115 (N108-I115).

10. The soluble CTLA4 mutant molecule of claim 9, wherein the mutation is a substitution of any of the amino acids in N108-I115 with a different amino acid selected from a group consisting of alanine, arginine, asparagine, aspartic acid, cysteine, glutamine, glutamic acid, glycine, histidine, isoleucine, leucine, lysine, methionine, phenylalanine, proline, serine, threonine, tryptophan, tyrosine, and valine.

11. The soluble CTLA4 mutant molecule of claim 1 further comprising an amino acid sequence which alters the solubility, affinity or valency of the soluble CTLA4 mutant molecule for binding to CD80 or CD86.

12. The soluble CTLA4 mutant molecule of claim 14, wherein the amino acid sequence comprises a human immunoglobulin constant region.

13. A nucleic acid molecule comprising a nucleotide sequence encoding the amino acid sequence corresponding to the soluble CTLA4 mutant molecule of claim 1.

14. A vector comprising the nucleotide sequence of claim 13.

15. A host vector system comprising the vector of claim 14 in a suitable host cell.

16. The host vector system of claim 15, wherein the suitable host cell is a prokaryotic cell or a eukaryotic cell.

17. A method for producing a soluble CTLA mutant protein comprising growing the host vector system of claim 16 so as to produce the protein in the host cell and recovering the protein so produced.

18. A soluble CTLA mutant protein produced by the method of claim 17.

19. A method for regulating a T cell interaction with a CD80 and/or CD86 positive cell comprising contacting the CD80 and/or CD86 positive cell with the soluble CTLA4 mutant molecule of claim 1 so as to regulate the T cell interaction.

20. The method of claim 19, wherein the soluble CTLA4 mutant molecule is any of L104EA29L, L104EA29T, or L104EA29W.

21. The method of claim 19, wherein the CD80 and/or CD86 positive cell is an antigen presenting cell.

22. The method of claim 19, wherein the interaction of the CTLA4-positive T cells with the CD80 and CD86 positive cells is inhibited.

23. A method for treating immunoproliferative diseases mediated by T cell interactions with B7 positive cells comprising administering to a subject the soluble CTLA4 mutant molecule of claim 1, in an amount effective to regulate T cell interactions with said B7 positive cells.

24. The method of claim 23, wherein said T cell interactions are inhibited.

25. The method of claim 23, wherein the immunoproliferative disease is graft versus host disease.